“PREDICTION OF NEW-ONSET ATRIAL FIBRILLATION BY ATRIAL TISSUE DOPPLER IMAGING - A NOVEL METHOD TO MEASURE TOTAL ATRIAL CONDUCTION TIME”

Dissertation submitted for

D.M. DEGREE EXAMINATION

BRANCH II – CARDIOLOGY

MADRAS MEDICAL COLLEGE

AND

GOVERNMENT GENERAL HOSPITAL

CHENNAI – 600 003

THE TAMIL NADU DR.M.G.R MEDICAL UNIVERSITY

CHENNAI – 600 032

AUGUST 2010

"Learn to heal"

CERTIFICATE

This is to certify that the dissertation entitled "**PREDICTION OF NEW-ONSET ATRIAL FIBRILLATION BY ATRIAL TISSUE DOPPLER IMAGING - A NOVEL METHOD TO MEASURE TOTAL ATRIAL CONDUCTION TIME"** is the bonafide original work of **DR.C.R.MADHU PRABHUDOSS** in partial fulfillment of the requirements for D.M. Branch-II (CARDIOLOGY) examination of THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY to be held in August 2010. The period of post-graduate study and training was from July 2007 to July 2010.

THE DEAN

MADRAS MEDICAL COLLEGE &
GOVERNMENT GENERAL HOSPITAL
CHENNAI - 600 003.

PROF.GEETHA SUBRAMANIAN M.D., D.M,
FIAE, FISE, FCSI, FISC.
PROFESSOR AND HEAD OF THE
DEPARTMENT OF CARDIOLOGY
MADRAS MEDICAL COLLEGE &
GOVERNMENT GENERAL HOSPITAL
CHENNAI - 600 003.

DECLARATION

I **DR.C.R.MADHU PRABHUDOSS,** solemnly declare that this dissertation entitled, "**PREDICTION OF NEW-ONSET ATRIAL FIBRILLATION BY ATRIAL TISSUE DOPPLER IMAGING - A NOVEL METHOD TO MEASURE TOTAL ATRIAL CONDUCTION TIME"**is a bonafide work done by me at the department of Cardiology, Madras Medical College and Government General Hospital during the period 2007 – 2010 under the guidance and supervision of the Professor and Head of the department of Cardiology of Madras Medical College and Government General Hospital, Professor **Geetha Subramanian M.D.D.M, FIAE, FISE, FCSI, FISC.** This dissertation is submitted to The Tamil Nadu Dr.M.G.R Medical University, towards partial fulfillment of requirement for the award of **D.M. Degree (Branch-II) in Cardiology.**

Place : Chennai

Date: 26-05-2010

DR.C.R.MADHU PRABHUDOSS

ACKNOWLEDGEMENTS

A great many people made this work possible. I thank my Dean for allowing me to conduct this study.

My warmest respects and sincere gratitude to our beloved ***Prof Geetha Subramanian*** *Professor and Head of the Department of Cardiology, Government General Hospital, Chennai. But for his constant guidance this study would not have been possible.*

I am indebted to ***Prof. Geetha Subramanian, Prof. B.Ramamurthy, Prof.M.Somasundaram, Prof. P.Arunachalam and Prof. V.E.Dhandapani*** *without whom, much of this work would not have been possible.*

I acknowledge ***Dr M.A.Rajasekar*** *for the many useful comments he made during this project.*

*In addition, I am grateful to****, Dr.G.Gnanavelu, Dr.S.Venkatesan, Dr.G.Ravishankar, Dr.JustinPaul, Dr.C.Elangovan Dr.S.Raghothaman and Dr.G.Prathapkumar for*** *tracing all those waveforms and guidance.*

Last but not the least I thank all my patients for their kind cooperation.

Contents

INTRODUCTION

Atrial fibrillation constitutes the commonest cardiac rhythm disorder and is the major determinant of Outcome in valvular, myocardial, and ischemic heart disorders. There is no simple tool available to predict the onset this arrhythmia.

Electrophysiological parameters are too complex and can not be obtained in bedside.

Tissue Doppler analysis of atrial muscle gives us an opportunity to measure the critical total atrial conduction time non invasively**.**

AIM

To predict new-onset Atrial Fibrillation (AF) by measuring the total atrial conduction time by atrial tissue Doppler imaging.

REVIEW OF LITERATURE

HISTORY OF ECHOCARDIOGRAPHY

Many histories of diagnostic ultrasound[9] and cardiac ultrasound in particular, have been written. They all seem to address this field from a different perspective. One can begin the history in the twentieth century, Roman times, or any of the centuries in between. It is stated that a Roman architect, Vitruvius, first coined the word echo. A Franciscan friar, Marin Mersenne (1588-"1648), is frequently called the father of acoustics because he first measured the velocity of sound. Another early physicist, Robert Boyle (1627-1691), recognized that a medium was necessary for the propagation of sound. Abbe Lazzaro Spallanzani (1727-1799) is frequently referred to as the father of ultrasound. He demonstrated that bats were blind and in fact navigated by means of echo reflection using inaudible sound. In 1842, Christian Johann Doppler (1803-1853) noted that the pitch of a sound wave varied if the source of the sound was moving. He worked out the mathematical relationship between the pitch and the relative motion of the source and the observer. The ability to create ultrasonic waves came in 1880 with the discovery of piezoelectricity by Curie and Curie. They noted that if certain crystalline materials are compressed, an electric charge is produced between the opposite surfaces. They then noted that the reverse was also true. If an electrical potential is applied to a crystal, it is compressed and decompressed depending on the polarity of the electric charge, and thus very high frequency sound can be produced. In 1912, a British engineer, L. F. Richardson, suggested that an echo technique could be used to de- tect underwater objects. Later during World War I, Paul Langevin was given the duty of

detecting enemy submarines using sound, which culminated in the development of sonar. Sokolov described a method for using reflected sound to detect metal flaws in 1929. In 1942, Floyd Firestone, an American engineer, began to apply this technique and received a patent. It is this flaw detection technique that ultimately was used in medicine.

An Austrian, Karl Dussik, was probably the first to apply ultrasound for medical diagnosis in 1941. He initially attempted to outline the ventricles of the brain. His approach used transmission ultrasound rather than reflected ultrasound. After World War II, many of the technologies developed during that war, including sonar, were applied for peaceful and medical uses. In 1950, W. D. Keidel, a German investigator, used ultrasound to examine the heart. His technique was to transmit ultrasonic waves through the heart and record the effect of ultrasound on the other side of the chest. The purpose of his work was to try to determine cardiac volumes. The first effort to use pulse-reflected ultrasound, as described by Firestone, to examine the heart was initiated by Dr. Helmut Hertz of Sweden. He was familiar with Firestone's observations and in 1953 obtained a commercial ultrasonoscope, which was being used for nondestructive testing. He then collaborated with Dr. Inge Edler who was a practicing cardiologist in Lund, Sweden. The two of them began to use this commercial ultrasonoscope to examine the heart. This collaboration is commonly accepted as the beginning of clinical echocardiography as we know it today.

The original instrument[9] was quite insensitive. The only cardiac structures that they could record initially were from the back wall of the heart. In retrospect, these echoes probably came from the posterior left ventricular wall. With some modification of their instrument, they were able to record an echo from the anterior leaflet of the mitral

valve. However, they did not recognize the source of this echo for several years and originally attributed the signal to the anterior left atrial wall. Only after some autopsy investigations did they recognize the echo's true origin. Edler went on to perform a number of ultrasonic studies of the heart. Many of the cardiac echoes currently used were first described by him. However, the principal clinical application of echocardiography developed by Edler was the detection of mitral stenosis. He noted that there was a difference between the pattern of motion of the anterior mitral leaflet in patients who did or did not have mitral stenosis. Thus, the early studies published in the mid-1950s and early 1960s primarily dealt with the detection of this disorder.

The work being done in Sweden was duplicated by a group in Germany headed by Dr. Sven Effert. Their publications began to appear in the late 1950s and were primarily duplications of Edler's work describing mitral stenosis. One notable observation made by Effert and his group was the detection of left atrial masses. Schmitt and Braun in Germany also began working with ultrasound cardiography and published their work in 1958, again repeating what Edler and Effert had been doing. Edler and his co-workers developed a scientific film that was shown at the Third European Congress of Cardiology in Rome in 1960. Edler et al. also wrote a large review of cardiac ultrasound as a supplement to Acta Medica Scandinavica, which was published in 1961, and remained the most comprehensive review of this field for more than 10 years. In the movie and the review, Edler and his co-investigators described the ultrasonic techniques for the detection of mitral stenosis, left atrial tumors, aortic stenosis, and anterior pericardial effusion.

Despite their initial efforts at using ultrasound to examine the heart, neither Edler nor Hertz really anticipated that this technique would flourish. Helmut Hertz was primarily interested in being able to record the ultrasonic signals. In the process, he developed ink jet technology and only spent a few years in the field of cardiac ultrasound. He devoted most of the rest of his career to ink jet technology, for which he held many important patents. He also advised Siemens Corporation, who provided their first ultrasonic instrument, that they should not enter the field of cardiac ultrasound because he personally did not feel that there was a great future in this area (Effert, personal communication, 1996). Edler too did not develop any further techniques in cardiac ultrasound. He retired in 1976 and until then was primarily concerned with the application of echocardiography for mitral stenosis and, to a lesser extent, mitral regurgitation. He never became involved with any of the newer techniques for pericardial effusion or ventricular function.

DOPPLER TISSUE IMAGING[9]

Routine Doppler imaging typically targets blood flow and hence the receiver characteristics, including the frequency filters that determine the range of velocities to be interrogated, are set to maximize the shifts anticipated with moving blood and to exclude the velocity shifts that would be seen with slower moving structures. Because red blood cells are relatively weak reflectors and tissue is a fairly intense reflector, filters are also adjusted to exclude highly reflective objects and to maximize less reflective objects when using conventional Doppler. Doppler tissue imaging uses the same principles; however, the target is tissue rather than red blood cells. For this purpose, filters are set to

parameters opposite those needed to accurately detect red blood cell motion. Because tissue has a greater reflectivity and slower motion, instrumentation filters are set to exclude high velocities and low-intensity reflectors. With this technique, either the myocardium or fibrous skeleton of the heart can be targeted and weaker reflections from the higher velocity blood cells relatively excluded.

One of the initial applications of this technique was to use color flow imaging display methodology and to saturate an area of interest with Doppler interrogation. The color Doppler signal from the moving tissue was then superimposed on the two-dimensional gray-scale image. Using traditional blue-red encoding for direction of motion, this results in dissimilar color encoding even when walls are moving at a similar velocity. The normally moving ventricular septum will be encoded in blue (motion away from the transducer), and the normal anterior motion of the posterior wall will be encoded in red. This results in opposite color encoding of opposing walls, which each have normal directional motion. The color encoding scheme can be changed to be unidirectional, recording only velocity irrespective of direction, in the same color. This has the disadvantage of encoding a dyskinetic wall with the same color as a normally contracting wall. This technique has substantial potential in that it would allow superimposing information regarding velocity and direction of motion on the anatomic image. It is limited in its applicability by relatively low frame rates and the inability to fully saturate the signal. Other limitations include potential aliasing of the color signal with higher velocity motion and limited registration of velocity information at an angle of incidence ($\hat{I}_{,}$) exceeding 30 degrees. Signal-to-noise ratios are typically relatively low,

and in poor-quality images, there may be substantial bleeding of the color signal from the tissue into the blood pool.

The initial attempts at color encoding and Doppler tissue imaging used standard Doppler shifts of velocity. The Doppler signal in the power or energy domain can also be encoded. The power domain refers to the registration of the intensity or amplitude of the reflected Doppler shifts rather than just their velocity. In theory, this format may be of benefit because of its higher signal-to-noise ratio and has been used fairly extensively in contrast echocardiography.

Complete saturation of the image with color signal has seen little clinical acceptance because of the limitations of frame rate and saturation with an adequate signal-to-noise ratio. It should be recognized that the original source signal is actually pulsed Doppler information, acquired from a wide area of interest and targeted to tissue motion. A greater degree of acceptance has been seen by displaying the spectral signal of velocity and extracting quantitative information from localized areas using this technique. Analogous to the use of a sample volume in blood flow imaging, a sample volume can be placed within the myocardium or mitral or tricuspid anulus and the direction and velocity of the myocardium at that point in space accurately determined.

A variation on Doppler tissue imaging is to acquire color-encoded images of tissue motion along an M-mode interrogation line. This represents a combination of M-mode echocardiography, color Doppler imaging, and quantitative Doppler tissue imaging. Color tissue Doppler M-mode imaging is a high temporal and spatial resolution technique for investigating myocardial mechanics, and the information thus extracted can

secondarily be employed for determining velocity gradients between adjacent points or more recently for strain rate imaging.

As Doppler imaging inherently detects motion and determines velocity, this is the fundamental parameter available. From this displacement of the tissue, the distance traveled can be determined as the product of velocity and time. More complex derivatives of the velocity determination include the calculation of the velocity gradient between two points and strain or strain rate imaging, which are measures of tissue deformation. The simplest calculation that can be derived from two-point analysis is the absolute difference in velocities. This has had clinical applicability in determining the gradient between endocardial and epicardial velocities, which may be a more sensitive indicator of myocardial ischemia than an absolute decrease in velocity across the entire myocardial wall.

More complex derivatives of these measurements include strain and strain rate, both of which are more load independent than parameters such as ejection fraction and wall motion and provide a more direct assessment of myocardial contractility. Because strain rate refers to the first derivative of motion (i.e., velocity change), it is more directly derived from Doppler tissue imaging, which inherently is a velocity calculation. strain is defined as the change in distance between two points divided by the initial length (L_0). Mathematically, it is a unitless number. Strain rate is the first derivative of strain and is calculated as the change in velocity between two points divided by the distance (L) between the two points. Mathematically, the units of strain rate are1/s.

Displacement, strain, and strain rate can be displayed in several formats. The easiest to understand is a graphic output of either of these parameters over time. One or more discrete regions of interest can be identified and simultaneously graphed for comparison of displacement, strain, or strain rate at any given time point. Because these parameters are derived from tissue Doppler imaging, which has high spatial resolution, it is often beneficial to simultaneously display these values for multiple points throughout the ventricular myocardium or for all points along a continuous line drawn through the myocardium. Display of this type of information requires a different format than a simple x/y graph as is appropriate for a single point. M-mode display in which time is conventionally displayed on the x axis, distance around the ventricle on the y axis, and the value of displacement, strain, or strain rate in color. This is analogous to the display of Doppler velocity data in a color M-mode but obviously incorporates the third dimension of location within the heart. Numerous studies have demonstrated that Doppler imaging derived myocardial gradients as well as strain and strain rate provide a higher resolution evaluation of myocardial mechanics than evaluation of wall motion, myocardial thickening, or tissue velocities alone.

Tissue Doppler imaging (TDI) [8] is a novel use of ultrasound to image the motion of tissue with Doppler echocardiography. Doppler echocardiography records and displays the velocities of the moving targets. When Doppler echocardiography is used to measure blood flow velocity, erythrocytes are the targets. Their normal velocity ranges from 10 cm/s in the venous circulation to 150 cm/s in the arterial circulation. However, the velocities of myocardial tissue are much lower (1-20 cm/s), but their amplitudes are

greater than those produced by blood. Therefore, Doppler ultrasound instruments high frame rate. A special function key needs to be selected to activate TDI. After TDI has been selected, the subsequent operation is identical to that used to perform regular pulsed wave Doppler echocardiography, except that the TDI gain needs to be lowered from the regular gain setting used for blood flow Doppler recordings and the velocity scale needs to be adjusted to a lower aliasing velocity (about 20-30 cm/s or even lower) to optimize TDI signals. Some ultrasound units adjust these variables automatically when the TDI function is selected. Also, TDI can be displayed in the color mode, just as in color imaging of blood flow. Tissue velocities are color-coded by autocorrelation: red for tissue moving toward the transducer and blue for tissue moving away from the transducer. Movement and velocities of cardiac structures are regulated by the underlying systolic function and diastolic function of the heart. Isaaz and colleagues were able to obtain a pulsed Doppler profile of the left ventricular (LV) posterior wall. This was expanded by McDicken and colleagues (who developed a prototype of color Doppler velocity display of myocardial wall dynamics). Currently, TDI is an integral part of an echocardiography examination in the areas discussed in the following sections.

ASSESSMENT OF MYOCARDIAL RELAXATION[8]

Early diastolic velocity (Ea) of the mitral anulus measured with TDI is a good indicator of LV myocardial relaxation. This is one of the most important components of myocardial diastolic function the mitral anulus can be appreciated visually from the parasternal long-axis and apical four-chamber views, but TDI records and demonstrates the velocity of the longitudinal motion in numerical value. In the normal

heart with normal myocardial relaxation, Ea increases with an increasing transmitral gradient, increasing preload, exercise, and dobutamine infusion . However, when myocardial relaxation is impaired because of aging or a disease process, Ea is affected less or even unchanged by preload or transmitral gradient. Velocities of longitudinal mitral anulus motion are best obtained from apical views. Although various locations of the mitral anulus can be interrogated with TDI, the two most frequently used locations are the septal (or medial) and lateral mitral anulus. Usually, Ea from the lateral anulus is higher (normally >15 cm/s) than that from the medial anulus (normally >10 cm/s). In our laboratory, mitral anulus velocities are usually, but not always, obtained from the septal anulus. Regional myocardial dysfunction or valvular surgery involving the mitral anulus may affect mitral anulus velocities. A localized disease process, such as lateral myocardial infarction, can result in mitral anulus velocities being lower at the lateral anulus than at the septal anulus.

Table 1

Comparison of two-dimensional (2D) gray scale and tissue doppler imaging (TDI)

		TDI	
Variable	**2D Gray Scale**	**Color Doppler**	**Pulsed Doppler**
Temporal resolution	30-50 frames/s (but 400 frames/s possible)	20-90 frames/s >150 frames/s possible)	>250 frames/s
Spatial resolution	1 ×1 mm (may be less)	Typically 2 × 2 mm	1 ×1 mm
Angle dependency	No	Yes	Yes
Applicability to all myocardium	++	+++	+++
Intramural function	No	Yes	No
Attenuation dependency	++++	++	+

Late diastolic velocity (Aa) of the mitral anulus[8] at the time of atrial contraction increases during early diastolic dysfunction, as is the case for the mitral inflow A wave, but decreases as atrial function deteriorates. Aa has been correlated with left atrial (LA) function.

ESTIMATION OF LEFT VENTRICULAR FILLING PRESSURE[8]

LV diastolic filling pressures can be estimated reliably with 2D and Doppler echocardiography. The deceleration time (DT) of mitral inflow early diastolic velocity (E) has a good inverse correlation with the pulmonary capillary wedge pressure (PCWP) of less than 130 milliseconds usually indicate a PCWP greater than 20 mm Hg. However, mitral inflow DT alone is not highly accurate in patients who have a relatively normal LVEF or atrial fibrillation. Because Ea is reduced in patients with impaired relaxation and is affected less by preload than mitral inflow E, the ratio (E/Ea) between mitral inflow early diastolic velocity and mitral anulus early diastolic velocity increases as PCWP increases. Investigations at Baylor College and in our laboratory have demonstrated that PCWP is higher than 20 mm Hg when E/Ea is more than 10 (using the lateral anulus Ea) or 15 (using the medial anulus Ea). This ratio works well even in patients who have fused mitral inflow signals, preserved LVEF, and atrial fibrillation. The only exception is patients with constrictive pericarditis, in whom Ea, especially from the medial anulus, is increased (>8 cm/s) and E/Ea is reduced with high filling pressures.

Because PCWP can be estimated reliably with E/Ea, estimation of PCWP with exercise is feasible (which is helpful is assessing patients who have exertional dyspnea).

EVALUATION OF REGIONAL AND GLOBAL SYSTOLIC FUNCTION

The extent of systolic movement of the mitral anulus correlates with LV systolic function and stroke volume. Normally, the systolic velocity (Sa) of the mitral anulus is more than 6 cm/s. Although TDI of the mitral anulus reflects the global systolic and diastolic function of the LV, segmental or regional function can be assessed by performing TDI of various LV segments by placing the sample volume (2-5 mm) in the region of interest. The size of the sample volume depends on the location and intensity of the signal and is usually between 2 and 5 mm. Further clinical experience with this variable will determine if Sa can replace other more commonly used systolic variables.

TISSUE VELOCITY GRADIENT

TDI can measure the difference in velocities of adjacent myocardial tissues (velocity gradient), and this can be used to assess the viability and deformation (strain) of the myocardium. The velocity of the endocardium is normally higher than that of the epicardium, thus producing a tissue velocity gradient. In akinetic but viable or nontransmurally infarcted myocardium, the myocardial velocity gradient persists, but there is no velocity gradient in scarred or transmurally infarcted myocardium. Because days to weeks are needed for myocardial contractility to recover after successful

reperfusion of an occluded coronary artery, measurement of the tissue velocity gradient can be useful in patients with an acute myocardial infarction. To record or display the myocardial velocity gradient, the direction of myocardial contractility needs to be aligned in parallel with the direction of the ultrasound beam. Therefore, imaging views are limited to the parasternal windows to image anterior or posterior walls.

CARDIAC TIME INTERVALS

Cardiac time intervals are regulated precisely by the mechanics and functions of the myocytes; hence, these intervals are a good measure of cardiac function. TDI is well suited for determining the timing of myocardial events. The precise timing of these events is helpful in understanding the mechanism of myocardial relaxation and myocardial suction during early diastolic filling. In healthy hearts, in which efficient myocardial relaxation is used effectively to suck blood from the LA into the LV during early diastole, the time of onset of mitral inflow (E) coincides with that of myocardial early diastolic motion (relaxation) of the mitral anulus (Ea). However, in hearts with delayed myocardial relaxation and increased filling pressure, diastolic filling (onset of the E wave) depends more on the increased LA pressure and occurs earlier than the onset of the early diastolic motion of the mitral anulus (Ea). Therefore, the time interval between the onset of mitral E velocity and that of the mitral anulus diastolic motion (Ea) increases, and this increased interval has been proposed as a new variable to assess LV filling pressures.

A limitation of measuring cardiac time intervals by pulsed wave Doppler echocardiography is nonsimultaneity because different cardiac cycles are usually needed to measure various intervals which in turn are used together. One solution is to have the capability of obtaining multiple pulsed wave Doppler recordings simultaneously. Another creative means to measure cardiac intervals from a single cardiac cycle is to use tissue Doppler anatomic color M-mode from the anterior mitral leaflet. From this technique, isovolumic contraction time, isovolumic relaxation time, and LV ejection time can be measured reliably from a single cardiac cycle

Mechanical dyssynchrony is measured by time intervals between peak ejection systolic velocities or peak strain of multiple myocardial segments, as discussed below.

EVALUATION OF THICK WALLS

The ventricular walls become thick for several reasons including LV hypertrophy, hypertrophic cardiomyopathy, infiltrative cardiomyopathy, restrictive cardiomyopathy, and the athletic heart. These entities can usually be differentiated on the basis of clinical and laboratory findings, but differentiating them can occasionally be difficult. The evaluation of myocardial relaxation with TDI is able to distinguish between a thick athletic normal heart and other disease conditions (16). Mitral anulus motion is well preserved in the athletic heart because myocardial relaxation is preserved, but it is reduced in all other conditions that have impaired myocardial relaxation.

PROGNOSTICATION

Because E/Ea can estimate LV filling pressures and patients with increased filling pressures have higher rates of morbidity and mortality, it is expected that a high E/Ea predicts a poor outcome. E/Ea more than 15 was found to be associated with increased mortality of patients with acute myocardial infarction . By itself, Ea is also a good predictor for clinical outcome. In various clinical conditions, patients who have an Ea less than 5 cm/s are more likely to have a much higher mortality than those with an Ea more than 5 cm/s.

STRAIN AND STRAIN RATE IMAGING

Myocardial velocities measured with TDI may be overestimated or underestimated by translational motion or tethering of the myocardium, respectively. This limitation can be overcome by measuring the actual extent of myocardial deformation (stretching or contraction) by strain (Îµ) and strain rate imaging (Fig. 5-3). Strain rate is the rate of change in length calculated as the difference between two velocities normalized to the distance between them; it is expressed as $seconds^{-1}$. By convention, shortening is represented by negative values and lengthening by positive values for both strain and strain rate:

Strain rate = $(V_a - V_b)/d$

Where V_a - V_b is the instantaneous velocity difference at points a and b, and d is the distance between the two points.

Strain (ε) is the percentage change in length during myocardial contraction and relaxation and is expressed as a percentage:

$$\varepsilon = (L_{1-} L_0) / L_0$$

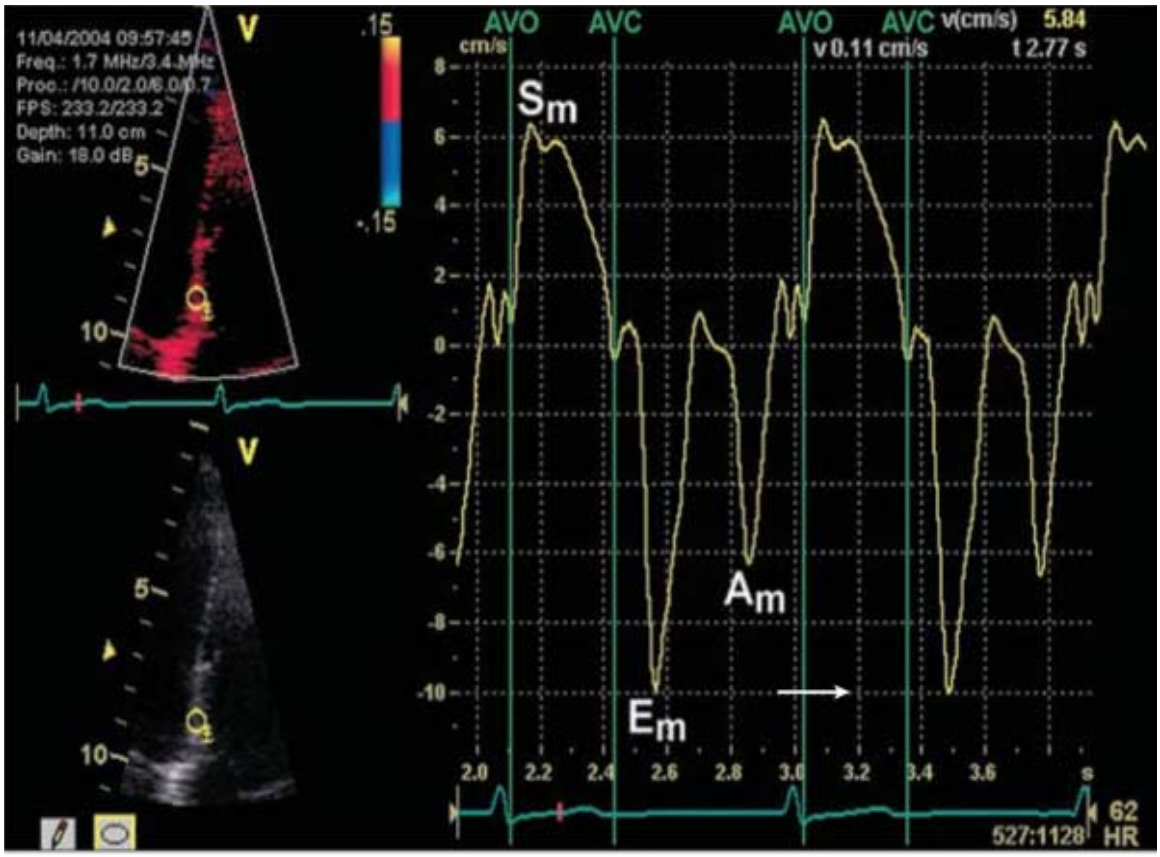
$$= \Delta L / L_0$$

where L0 is the original length, L_1 is the final length, and ΔL is the change in length.

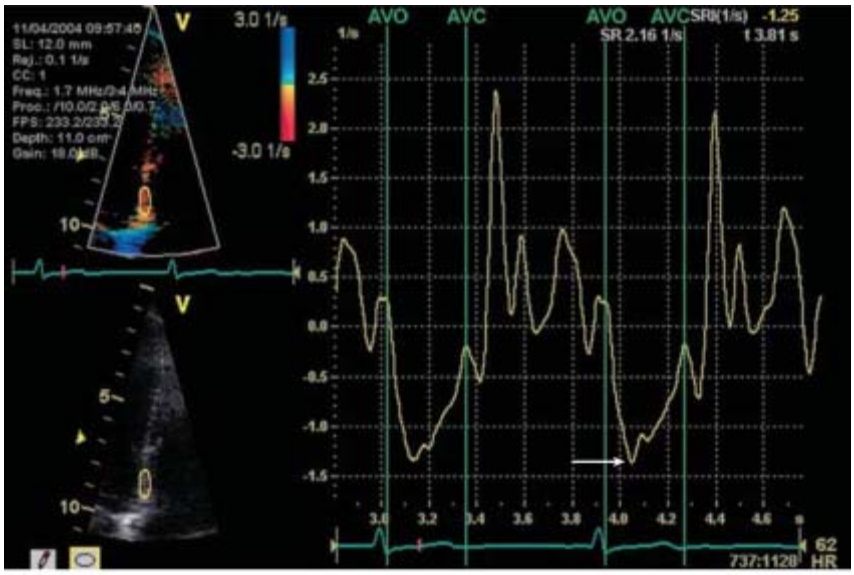
Strain can be derived echocardiographically by the following:

$$\varepsilon = \int^{t}_{\text{to SR.dt}}$$

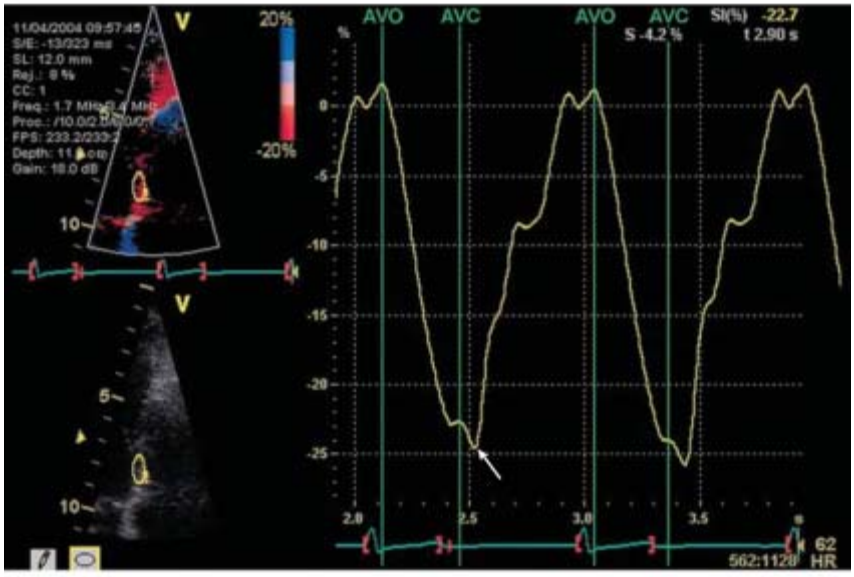
Where strain (ε) is the sum of the instantaneous strain rate (SR) values from starting time (t_0) to ending time (t).


11/04/2004 09:57:45
Freq.: 1.7 MHz/3.4 MHz
Proc.: /10.0/2.0/6.0/0.7
FPS: 233.2/233.2
Depth: 11.0 cm
Gain: 18.0 dB
V
.15
-.15
5
10
V
5
10
AVO
AVC
AVO
AVC
v(cm/s) 5.84
v 0.11 cm/s
t 2.77 s
cm/s
Sm
Am
Em
2.0
2.2
2.4
2.6
2.8
3.0
3.2
3.4
3.6
s
62
HR
527:1128

A



B



C

FIGURE 1A, Recording of tissue velocity. The sample volume was placed at the basal portion of the inferior septum. Peak systolic velocity (Sm) was slightly more than 6cm/sec, early diastolic velocity (Em) was 10cm/sec, and late diastolic velocity (Am) was 6cm/sec.

B, Recording of strain rate, which represents the rate of deformation; the peak negative strain rate (arrow) was -1.3/sec.

C, Strain recording, which is the integration of the strain rates; the negative peak strain (arrow) occurred slightly after aortic valve closure (AVC). The normal strain value is usually more than -15 percent. AVO= aortic valve opening.

Tissue tracking, also known as displacement, is similar to strain, except it is the integral of the tissue velocity over a given time. It represents the distance a region of interest moves relative to its original location.

In the normal heart, longitudinal strain rate values are similar from the base to the apex, unlike tissue velocity, which is higher at the base than at the apex. Every effort is made to ensure that the direction of tissue movement is less than 30 degrees from the direction of the beam, but this is technically challenging in the apical segments as the angle becomes wider. The narrow-sector angle approach on an individual wall obviates some of the above problems, which precludes concurrent comparison of contralateral segments.

Strain imaging is similar to measuring the myocardial velocity gradient, which is limited to analyzing the myocardium that contracts in the direction that is parallel with the ultrasound beam. However, better spatial resolution and a higher frame rate (up to 200 frames/s) in strain rate imaging allow simultaneous calculation of the strain rate of the myocardium within a selected sector, which can be color coded. A curved cursor can be placed along the entire circumference of the LV to analyze regional strain rate. However, accurate measurements of strain rate depend on properly aligning the ultrasound beam so it is parallel with the direction of myocardial motion.

SPECKLE TRACKING ECHOCARDIOGRAPHY

Speckle tracking is a method for quantifying myocardial motion in various planes using 2D images. Reflection, scattering, and interference of the ultrasound beam in the myocardial tissue produce a speckle formation. Myocardial regions with unique speckle patterns in the gray scale 2D image can be tracked from frame to frame

throughout the cardiac cycle. This allows assessment of LV rotational motion, often referred to as torsion or twist. The spiral shape of the LV myocardial fibers results in a complex three-dimensional (3D) torsion mechanism for systolic contraction and untwisting for diastolic relaxation. The LV myocardium consists of two layers. The subendocardial layer wraps around the LV cavity in the direction of a right-handed helix, and the subepicardial layer wraps around in the direction of a left-handed helix. When viewed from the LV apex, apical rotation is counterclockwise and basal rotation is clockwise during systole. An analogy for LV contraction is the motion of wringing out a wet towel with your hands. As the two hands twist the ends of the towel in opposite directions Speckle tracking is an alternative method for quantification of LV systolic, and potentially diastolic, function. It also is another method for measuring strain using 2D images instead of the TDI method described above. Speckle tracking does not have the limitation of angle dependence that TDI-derived strain measurements have.

TISSUE DOPPLER IMAGING (TDI) AND STRAIN IMAGING OF LEFT ATRIUM[3]

The haemodynamic function of the left atrium (LA) [3] primarily modulates the left ventricular (LV) filling through its three components: a reservoir component during ventricular systole, a conduit component during early ventricular diastole, and a booster pump component during late ventricular diastole. The change of the LA function in different phases can be assessed non-invasively by echocardiography, using not only conventional methods such as changes in LA area and volume, but also novel techniques such as tissue Doppler imaging (TDI) and strain imaging. Tissue Doppler imaging

quantifies regional tissue motion velocity whereas strain and strain rate represent the extent of local tissue deformation and its rate, respectively. These novel technologies have been validated for the assessment of both global and regional

LV function and have recently been applied to the evaluation of regional LA function. From an electromechanical perspective, echocardiographic parameters that assess LA mechanical function may provide a greater understanding of atrial performance and its relationship with ventricular function.

Assessment of left atrial and appendage function by tissue Doppler imaging [3].

Both spectral pulse TDI or two-dimensional colour-coded TDI can be applied to generate a myocardial velocity curve to assess a regional LA function, by placing a small sample volume at an atrial segment of interest, usually about 2 mm for measuring velocity and preferably not more than 12 mm of length for strain and strain rate, because of its thin-walled structure. Unlike the spectral pulse TDI that has a better temporal resolution but can measure only one segment at a time, colour-coded TDI images can be processed offline and offer simultaneous multi-segment analyses of velocities and other TDI-derived parameters, such as strain and strain rate. Thus, different LA walls with their corresponding levels from the mitral annulus can be compared and assessed, in particular by offline analysis of colour TDI, such as septal and lateral walls in an apical four-chamber view, anterior and inferior walls in a two-chamber view. The atrial myocardial velocity curve consists of three major deflections: ventricular systolic (Sa), early ventricular diastolic (Ea), and late ventricular diastolic (atrial contraction, Aa) waves. In addition, the three components in strain and strain-rate imaging can also be

readily identified. The Aa-wave has been regarded as a direct measure of regional active atrial contraction on the longitudinal axis, which might be less load dependent. The Sa and Ea waves may represent the passive expansion and emptying components of the LA function. The feasibility and reproducibility of TDI parameters, in particular the peak velocity (VAa) and peak strain rate (SRAa) of the active atrial contraction, have been demonstrated in previous studies, in which both the inter- and intra-observer variability for measuring the VAa were reported to be within 10%.Although the velocity data could be easily obtained in nearly all patients, strain rate measurements were only feasible in about 95% of patients due to the relatively higher noise-to-signal ratio

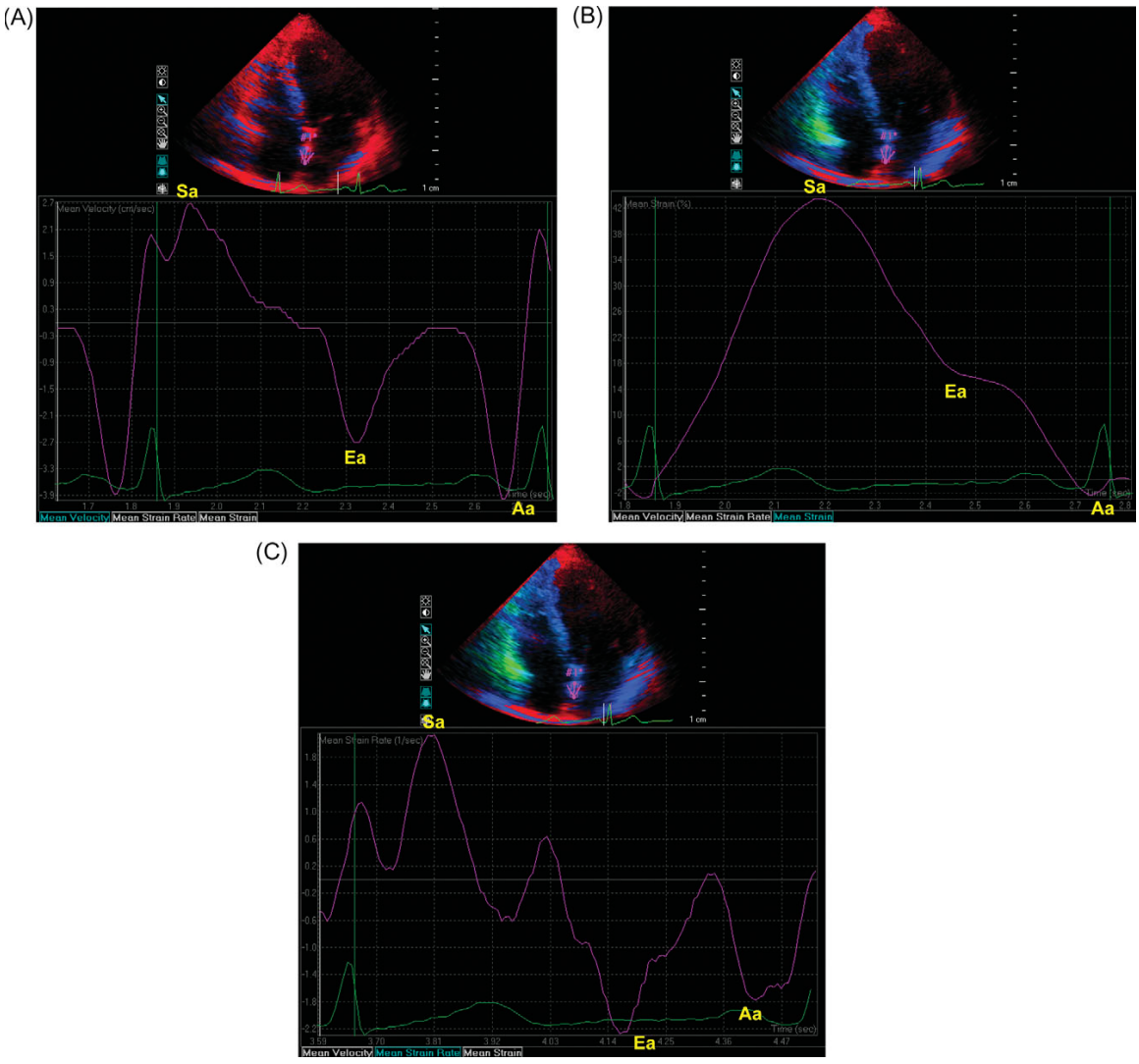


FIGURE 2 Assessment of regional atrial myocardial function by tissue Doppler imaging. The sample volume was placed at the mid-level of inter-atrial septum on colour tissue Doppler imaging image to reconstitute the myocardial velocity (A), strain (B), and strain rate (C) curves. They consist of ventricular systolic (Sa-wave), ventricular early diastolic (Ea-wave), and late diastolic (atrial contraction, Aa-wave) components

Left atrial appendage (LAA) [3] is a highly contractile structure with a pattern of contractions totally different from that of the LA main body. It is more compliant and therefore plays an important role in the LA reservoir function, especially during increases in the LA pressure or volume. Transoesophageal echocardiography (TEE) provides essential information about LAA structure and function. In addition to the LAA size, emptying and filling velocities, a characteristic triphasic TDI profile can be obtained readily at the tip, the septal, or lateral wall of the LAA by TEE. During sinus rhythm, there is an initial upward velocity in early atrial systole, i.e. in late ventricular diastole before the P-wave on ECG. This is followed by another upward velocity (after the P-wave), the late systolic wave (LSW), that has a higher amplitude and corresponds to the late emptying flow of the LAA. Lastly, the negative late diastolic wave (LDW) occurs during the LAA filling.

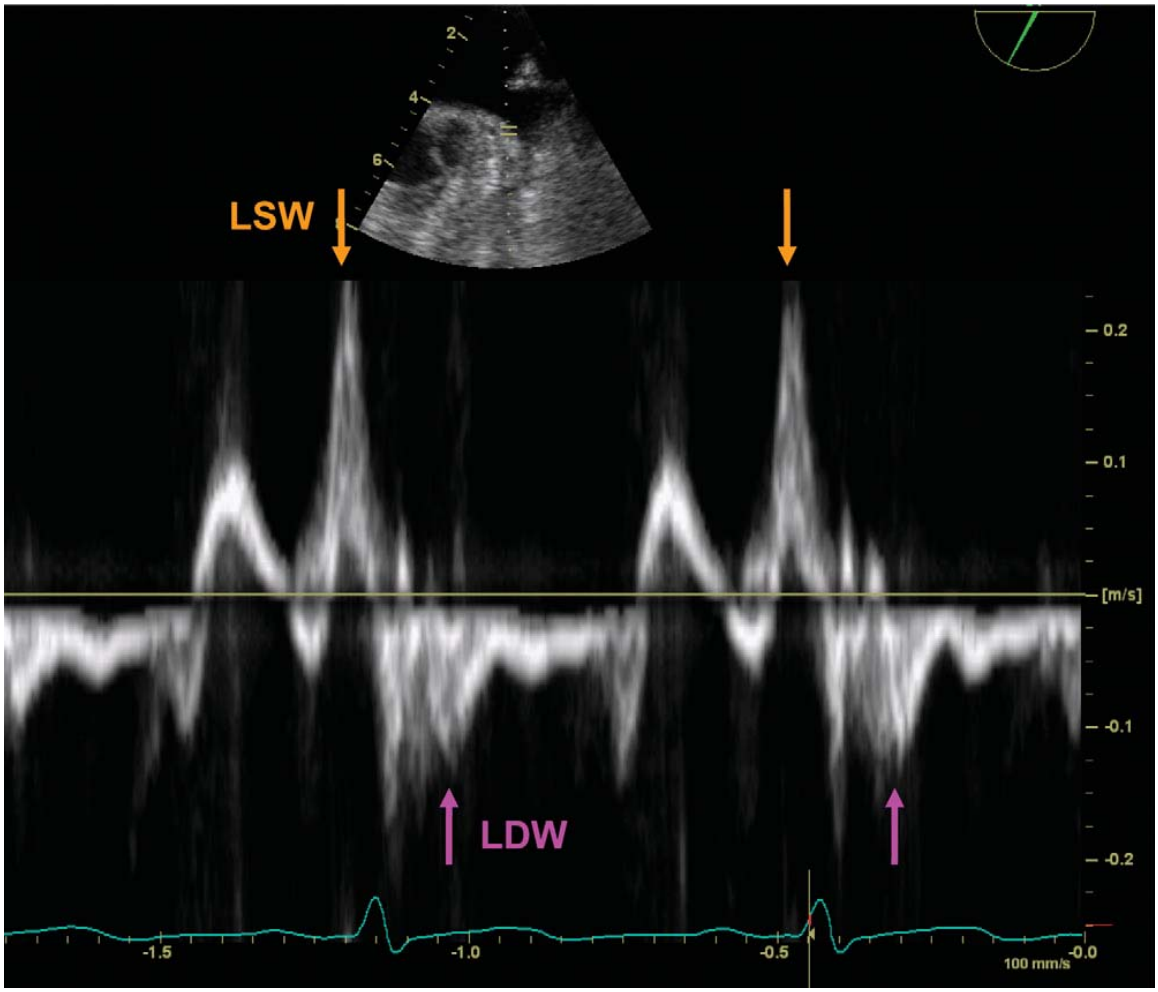


FIGURE 3 Myocardial performance of left atrial appendage. The sample volume was placed at the septal wall of left atrial appendage where the pulsed tissue Doppler imaging was recorded in transoesophageal echocardiography. The profile is normally triphasic, which includes the initial upward velocity before the P-wave on electrocardiogram, followed by another upward velocity with higher amplitude after the P-wave (late systolic wave, LSW) and the negative velocity (late diastolic wave, LDW).

Atrial fibrillation (AF) [1] is the most common sustained cardiac arrhythmia and may result in life-threatening complications such as stroke and heart failure. Unfortunately, treatment often comes too late—for example, when stroke is the first

manifestation of AF. Therefore, prediction and prevention of AF and its complications is essential. There has been an increase in the number of admissions to hospital for AF in recent years, demonstrating the need for primary prevention of new-onset AF. To enable primary prevention strategies, we have to identify which patients are at increased risk for the development of AF. Observational population studies resulted in the discovery of clinical and echocardiographic parameters that are associated with the development of AF. However, the currently available risk stratification parameters have limited predictive value in the individual patient. Age or underlying heart disease may result in atrial dilatation or a depressed intra-atrial conduction. This will lead to an increased total atrial conduction time (TACT) [1] and facilitate AF. Recently, we validated a new non-invasive echocardiographic method to determine the TACT, using transthoracic tissue Doppler imaging of the atria (the PA-TDI interval). We demonstrated that PA-TDI is an easy, fast and reliable method to estimate the TACT.

This study demonstrates that the TACT [1]as determined by tissue Doppler imaging may help to identify patients with a substrate vulnerable for AF. After correcting for possible confounders, the PA-TDI interval remains the most important predictor of new-onset AF. The longer the PA-TDI interval[1], the higher the incidence of new-onset AF. In fact, each 10 ms increase of PA-TDI is associated with an increased risk of developing AF of 37–52% in the next 2 years. Therefore, the PATDI interval may become a useful measure for risk stratification to improve efficiency of primary prevention of AF.

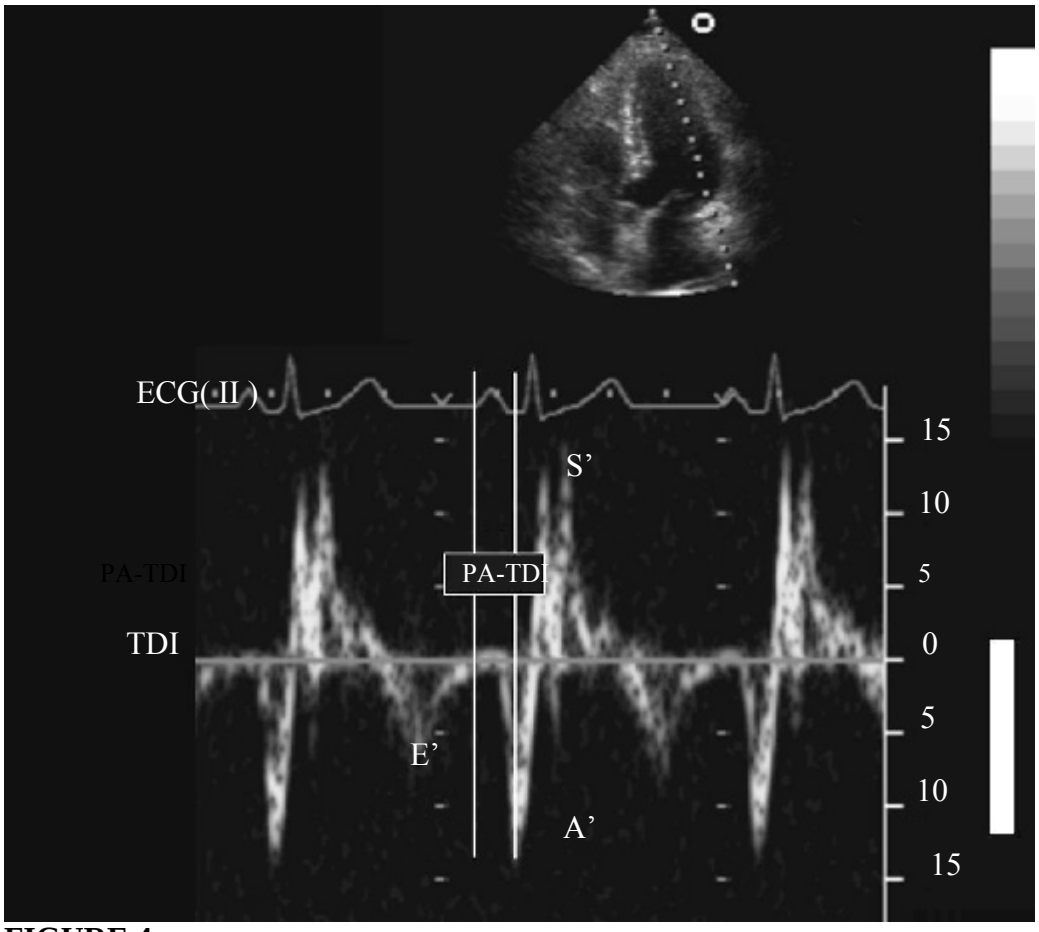


FIGURE 4

In the apical four-chamber view[1], the pulsed-wave tissue Doppler sample was placed on the lateral wall of the left atrium just above the mitral annulus. The PA-TDI interval, defined as the time-interval from initiation of the electrocardiographic P wave recorded by the echo machine (lead II) to the peak of the A' wave of the atrial tissue Doppler tracing (fig 4), was measured in three cardiac cycles and averaged.

ATRIAL FIBRILLATION

ELECTROCARDIOGRAPHIC RECOGNITION.

Atrial fibrillation is an arrhythmia characterized by seemingly disorganized atrial depolarizations without effective atrial contraction. It was once thought that all atrial fibrillation was caused by a single mechanism of multiple wavelets propagating in random fashion throughout the atria. It is now apparent that there are likely several mechanisms and that there may be some organization to atrial fibrillation. For example, in many patients, atrial fibrillation is caused by a focal discharge at rapid rates and fibrillatory conduction (heterogeneous conduction caused by rapidity of activation) through the atria. Nonetheless, all these potential mechanisms of atrial fibrillation have a common appearance on the ECG. During atrial fibrillation, electrical activity of the atrium can be detected on the ECG as small, irregular baseline undulations of variable amplitude and morphology, called f waves, at a rate of 350 to 600 beats/min. At times, small, fine, rapid f waves can occur and are detectable only by right atrial leads or by intracavitary or esophageal electrodes. The ventricular response is grossly irregular (irregularly irregular) and, in an untreated patient with normal AV conduction, is usually between 100 and 160 beats/min.

In patients with WPW syndrome, the ventricular rate during atrial fibrillation can at times exceed 300 beats/min and lead to ventricular fibrillation. Atrial fibrillation should be suspected when the ECG shows supraventricular complexes at an irregular rhythm and no obvious P waves. The recognizable f waves probably do not

represent total atrial activity but depict only the larger vectors generated by the multiple wavelets of depolarization that occur at any given moment.

Each recorded f wave is not conducted through the AV junction, so a rapid ventricular response comparable to the atrial rate does not occur. Many atrial impulses are concealed (not recorded in the ECG) because of a collision of wave fronts, or they are blocked in the AV junction without reaching the ventricles (i.e., concealed conduction, which accounts for the irregular ventricular rhythm). The refractory period and conductivity of the AV node are determinants of the ventricular rate. When the ventricular rate is very rapid or very slow, it may appear to be more regular. Even though conversion of atrial fibrillation to atrial flutter is accompanied by slowing of the atrial rate, an increase in the ventricular response can result, because more atrial impulses are transmitted to the ventricle as a result of less concealed conduction. Also, it is easier to slow the ventricular rate during atrial fibrillation than during atrial flutter with drugs such as digitalis, calcium antagonists, and beta blockers, because the increased concealed conduction makes it easier to produce an AV block.

CLASSIFICATION[11]

AF traditionally has been described as either paroxysmal or chronic. However, the definition of chronic varies greatly in the literature, often suggesting permanent AF. The American Heart Association (AHA), American College of Cardiology (ACC), and the European Society of Cardiology (ESC) have proposed a standardized classification scheme to describe AF, which we support. At the initial detection of AF, it may be difficult to be certain of the subsequent pattern of duration and

frequency of recurrences. Thus, a designation of first detected episode of AF is made on the initial diagnosis. When the patient has experienced two or more episodes, atrial AF is classified as recurrent. After the termination of an episode of AF, the rhythm can be classified as paroxysmal or persistent. Paroxysmal AF is characterized by self-terminating episodes that generally last <7 days (most <24 hours), whereas persistent AF generally lasts >7 days and often requires electrical or pharmacologic cardioversion. AF is classified as permanent when it has failed cardioversion or when further attempts to terminate the arrhythmia are deemed futile. It might be more appropriate to use the term established rather than permanent, because these patients can undergo successful ablation to restore and maintain sinus rhythm precluding the concept of permanent. Although this classification scheme is generally useful, the pattern of AF may change in response to treatment. Thus, AF that has been persistent may become paroxysmal during pharmacologic therapy with antiarrhythmic medications.

CLINICAL FEATURES [7].

Atrial fibrillation is a common arrhythmia that is found in 1 percent of persons older than 60 years to more than 5 percent of patients older than 69 years. From the Framingham data, the lifetime risk of developing atrial fibrillation after age 40 has been found to be 26.0 percent (95 percent confidence interval [CI], 24.0 to 27.0 percent) for men and 23.0 percent (21.0 to 24.0 percent). Estimates are that 2.2 million Americans have atrial fibrillation, which occurs more commonly in men than in women. A history of congestive heart failure, valvular heart disease and stroke, left atrial enlargement, abnormal mitral or aortic valve function, treated systemic hypertension, and advanced age are independently associated with the prevalence of atrial fibrillation. Most recently,

obesity has been found as an additional risk factor for developing atrial fibrillation. Four important aspects of atrial fibrillation are treatable contributing factors, control of the ventricular rate, prevention of recurrences, and prevention of thromboembolic episodes. Occult or manifest thyrotoxicosis should be considered in patients with recent-onset atrial fibrillation. Atrial fibrillation can be intermittent or chronic and may be influenced by autonomic activity. Atrial fibrillation, whether it is persistent or intermittent, predisposes to stroke. Symptoms as a result of atrial fibrillation are determined by multiple factors, including the underlying cardiac status, the rapidity and irregularity of ventricular rate, and loss of atrial contraction. Physical findings include a slight variation in intensity of the first heart sound, absence of 'a' waves in the jugular venous pulse, and an irregularly irregular ventricular rhythm. Often, with fast ventricular rates, a significant pulse deficit appears (i.e., the auscultated apical rate is faster than the rate palpated at the wrist, pulse deficit, because each contraction is not sufficiently strong to open the aortic valve or transmit an arterial pressure wave through the peripheral artery). If the ventricular rhythm becomes regular in patients with atrial fibrillation, conversion to sinus rhythm, atrial tachycardia, or atrial flutter with a constant ratio of conducted beats or the development of junctional tachycardia or VT should be suspected.

EMBOLIZATION AND ANTICOAGULATION [7].

In addition to hemodynamic alterations, the risk of systemic emboli, probably arising in the left atrial cavity or appendage as a result of circulatory stasis, is an important consideration. Nonvalvular atrial fibrillation is the most common cardiac disease associated with cerebral embolism. In fact, almost half of cardiogenic emboli in the United States occur in patients with nonvalvular atrial fibrillation. The risk of stroke

in patients with nonvalvular atrial fibrillation is five to seven times greater than that in controls without atrial fibrillation. Overall, 20 to 25 percent of ischemic strokes are caused by cardiogenic emboli. Many studies have evaluated the risk of stroke in patients with atrial fibrillation and the benefits of anticoagulation and antiplatelet therapy. Patients with mitral stenosis and atrial fibrillation have a 4 to 6 percent incidence of embolism per year. Risk factors that predict stroke in patients with nonvalvular atrial fibrillation include a history of previous stroke or transient ischemic attack (relative risk, 2.5), diabetes (relative risk, 1.7), history of hypertension (relative risk, 1.6), and increasing age (relative risk, 1.4 for each decade). Patients with any of these risk factors have an annual stroke risk of at least 4 percent if untreated. Patients whose only stroke risk factor is congestive heart failure or coronary artery disease have stroke rates approximately three times higher than patients without any risk factors. Left ventricular (LV) dysfunction and a left atrial size larger than 2.5cm/m2 on echocardiographic examination are associated with thromboembolism. Patients younger than 60 to 65 years of age who have a normal echocardiogram and no risk factors have an extremely low risk for stroke (1 percent per year). Therefore, the risk of stroke in patients with lone atrial fibrillation—that is, idiopathic atrial fibrillation in the absence of any structural heart disease or any of the risk factors discussed previously—is relatively low. A scoring system to predict risk of stroke accounting for all these clinical risks has been proposed from an analysis of the Framingham Heart Study.

The annual rate of stroke for the unanticoagulated control group in five large anticoagulation trials was 4.5 percent but was reduced to 1.4 percent (68 percent risk reduction) for the warfarin treated group (60 percent risk reduction in men; 84

percent risk reduction in women). Aspirin, 325 mg/day, produced a risk reduction of 44 percent. The annual rate of major hemorrhage was 1 percent for the control group, 1 percent for the aspirin group, and 1.3 percent for the warfarin group. No difference in stroke risk occurs between paroxysmal (intermittent) atrial fibrillation and sustained (chronic) atrial fibrillation. Anti-coagulation therapy is ap-proximately 50 percent more effective than aspirin therapy for the prevention of ischemic stroke in patients with atrial fibrillation.

Risk factors for anticoagulant-associated intracranial hemorrhage include excessive anticoagulation and poorly controlled hypertension. Elderly individuals are at increased risk for anticoagulant-associated brain hemorrhage, especially if over anticoagulated. From these and other data, it appears that individuals younger than 60 years of age without any clinical risk factors or structural heart disease (lone atrial fibrillation) do not require antithrombotic therapy for stroke prevention because of their low risk. The stroke rate is also low (less than 2 percent/year) in patients between the ages of 60 and 75 years with lone atrial fibrillation. These patients may be adequately protected from stroke by aspirin therapy. In very elderly (older than 75 years) patients with atrial fibrillation, anticoagulation should be used with caution and carefully monitored because of the potentially increased risk of intracranial hemorrhage. Nevertheless, elderly patients with atrial fibrillation are still likely to benefit from anticoagulation because they are at particularly high stroke risk. Food and drugs such as antibiotics and antiarrhythmics (e.g., amiodarone) can influence the effects of warfarin. Recommendations for antithrombotic therapy can be made. Any patient with atrial fibrillation who has risk factors for stroke (prior stroke or transient ischemic attack,

significant valvular heart disease, hypertension, diabetes, age older than 65 years, left atrial enlargement, coronary artery disease, or congestive heart failure) should be treated with warfarin anticoagulation to achieve an international normalized ratio (INR) of 2.0 to 3.0 for stroke prevention if the individual is a good candidate for oral anticoagulation. Patients with contraindications to anticoagulation and unreliable individuals should be considered for aspirin treatment. Patients with atrial fibrillation who do not have any of the preceding risk factors have a low stroke risk (2 percent/year or less) and can be protected from stroke with aspirin. In patients older than 75 years, anticoagulation should be used with caution and monitored carefully to keep the INR less than 3.0 because of the risk of intracranial hemorrhage.

The risk of embolism following cardioversion to sinus rhythm in patients with atrial fibrillation varies from 0 to 7 percent, depending on the underlying risk factors. This risk is independent of the mode of cardioversion, either by chemical (drug) or direct current (DC) shock. Patients at high risk are those with prior embolism, a mechanical valve prosthesis, or mitral stenosis. Low-risk patients are those younger than 60 years without underlying heart disease. The high-risk group should receive chronic anticoagulation (see later), regardless of whether they will undergo cardioversion. Patients not in the low-risk group who have atrial fibrillation longer than 2 days should receive warfarin to achieve an INR of 2.0 to 3.0 for 3 weeks before elective cardioversion and for 3 to 4 weeks after reversion to sinus rhythm. An alternative strategy is to obtain a transesophageal echocardiogram to exclude the presence of an atrial thrombus. It appears that this technique predicts a group at low risk for the development of thromboembolism following cardioversion, provided that the patients are immediately treated with heparin

followed by therapeutic doses of warfarin. Anticoagulation with heparin has been recommended for emergency cardioversion when 3 weeks of anticoagulation or a transesophageal echocardiogram cannot be obtained. No matter which strategy is used, anticoagulation should be continued for at least 4 weeks following cardioversion, because atrial contractile function may not fully return until then. These suggestions must be individualized for a given patient. For example, patients at risk of trauma by virtue of occupation, participation in sports, and episodes of dizziness or syncope are at increased risk of bleeding if given anticoagulants and should probably not receive warfarin. Patients should be warned about taking any new drugs, such as nonsteroidal anti-inflammatory agents, or dietary changes with foods containing vitamin K such as leafy vegetables, if they are receiving warfarin. There are no data to suggest that conversion to sinus rhythm eliminates the risk of thromboembolism and thus this should not be the sole purpose for doing so. In fact, data from the Atrial Fibrillation Follow-Up Investigation of Rhythm Management (AFFIRM) trial have suggested that the strategy of maintaining sinus rhythm is insufficient alone to prevent thromboembolism, and patients who are treated with a strategy of maintaining sinus rhythm should be continued on anticoagulation unless there is a contraindication.[12] Many patients can have asymptomatic recurrences of atrial fibrillation. Newer strategies for stroke prevention are being developed. Oral thrombin inhibitors (e.g., ximelagatran, melagatran) have been shown to be effective in preventing postoperative deep vein thrombosis and have been evaluated in a randomized trial for stroke prevention in atrial fibrillation. Although not yet approved for use, the potential advantage of these agents is their wider therapeutic

window not requiring monitoring, like warfarin. Left atrial occlusion, surgically or with a catheter-based system, is also being evaluated as a method for stroke prevention.

MANAGEMENT [7].

The goals of management of the patient with atrial fibrillation are to reduce the risk of thromboembolism (described earlier) and to control symptoms. The latter is accomplished by controlling the ventricular rate during atrial fibrillation and/or restoring and maintaining sinus rhythm. Currently, no clear benefit has been ascribed to one treatment strategy over the other (rate control versus rhythm control), particularly because antiarrhythmic drugs are only 50 to 70 percent effective and carry risks of proarrhythmia .Data from large clinical studies (e.g., AFFIRM, Pharmacologic Intervention in Atrial Fibrillation [PIAF], and Rate Control versus Electrical Cardioversion [RACE]) all have demonstrated that both treatment strategies are reasonable. In the largest of these studies, AFFIRM, 4060 patients with atrial fibrillation and risk factors for thromboembolism were randomized to either rhythm control or rate control treatment strategy, using standard pharmacological approaches. The study found no differences in mortality or quality of life between the two groups. In both groups, thromboembolic events occurred in those patients in whom anticoagulation was stopped. These studies compared treatment strategies and did not compare sinus rhythm to atrial fibrillation with rate control. In the AFFIRM trial, only 60 percent of the patients in the rhythm control arm were in sinus rhythm at follow-up, and patients were not rigorously monitored for atrial fibrillation recurrences between visits. The overall treatment strategy (i.e., ventricular rate control or restoration and maintenance of sinus rhythm) should be individualized for each patient and based on whether patients are

symptomatic from uncontrolled ventricular rates or from atrial fibrillation itself (i.e., loss of AV synchrony and atrial contraction) and risk for side effects from drugs. It can sometimes be difficult to determine whether a patient's symptoms are caused by rapid ventricular rates or the loss of atrial contraction. As a general rule, asymptomatic patients found to have atrial fibrillation on a routine ECG are not likely to require rhythm control, and rate control is usually sufficient. Ambulatory monitoring correlating the patient's ventricular response and rhythm to symptoms and exercise testing can be useful to this end. Trials of aggressive rate control or, conversely, cardioversion and maintenance of sinus rhythm are sometimes necessary to make this determination. Intolerance to medications or excessive risk in one strategy may necessitate switching strategies. As stated earlier, a rhythm control strategy should not be an alternative to anticoagulation therapy to reduce stroke risk. Many elderly patients tolerate atrial fibrillation well without therapy, because the ventricular rate is slow as a result of concomitant AV nodal disease. These patients often have associated sick sinus syndrome, and the development of atrial fibrillation represents a cure of sorts. Such patients may demonstrate serious supraventricular and ventricular arrhythmias or asystole after cardioversion, so the likelihood of establishing and maintaining sinus rhythm should be weighed against the risks of cardioversion or other forms of therapy.

ACUTE MANAGEMENT [7].

A patient with atrial fibrillation discovered for the first time should be evaluated for a precipitating cause, such as thyrotoxicosis, mitral stenosis, pulmonary emboli, or pericarditis. The patient's clinical status determines initial therapy, the objectives being to slow the ventricular rate and/or restore atrial systole. If sudden onset

of atrial fibrillation with a rapid ventricular rate results in acute cardiovascular decompensation, electrical cardioversion is the initial treatment of choice. For other patients, the decision to cardiovert is largely based on the individual clinical situation. The need to restore sinus rhythm must be weighed against the likelihood of successful cardioversion and long-term maintenance of sinus rhythm. Maintenance of sinus rhythm after cardioversion is influenced by the duration of atrial fibrillation and, in some adults, atrial dilation. Animal studies have indicated that atrial fibrillation begets atrial fibrillation—the longer the patient has atrial fibrillation, the greater the likelihood that it will remain because of a process called electrophysiological remodeling. Similar electrophysiological abnormalities can be demonstrated in patients following short episodes of atrial fibrillation, but the mechanism(s) and clinical significance remain unknown. Although parameters such as atrial size and duration of atrial fibrillation predict the success of cardioversion in population studies, enlarged atria and atrial fibrillation of long duration are not absolute contraindications to attempted cardioversion. Internal cardioversion via intracavitary catheters can be effective when transthoracic shocks fail, particularly in obese patients or in those with significant pulmonary disease. However, with the advent of biphasic external defibrillators, the success rates and the energy requirement for cardioversion have improved and the need to perform internal cardioversions has decreased. Alternatively, antiarrhythmic drugs that lower defibrillation thresholds such as ibutilide can be used to pretreat the patient and increase the success of DC cardioversion. Because atrial contraction may not return immediately after restoration of electrical systole, clinical improvement may be delayed. DC cardioversion establishes normal sinus rhythm in more than 90 percent of patients, but sinus rhythm remains for 12

months in only 30 to 50 percent. Patients with atrial fibrillation of less than 12 months' duration have a greater chance of maintaining sinus rhythm after cardioversion. For patients who do not require emergent cardioversion, chemical cardioversion with IV antiarrhythmic drugs is effective in 35 to 75 percent of patients, depending on the population studied. Although procainamide has been used extensively for years, no well-controlled studies have been performed to determine its efficacy. Outside the United States, IV flecainide has been used with good results. IV amiodarone appears to be less effective, with no difference in conversion rates from placebo. IV ibutilide is also effective in about 35 to 75 percent of patients, depending on the population studied. In the absence of decompensation, the patient can be treated with drugs such as digitalis, beta blockers, or calcium antagonists to maintain a resting apical rate of 60 to 80 beats/min that does not exceed 100 beats/min after slight exercise. The combined use of digitalis and a beta blocker or calcium antagonist can be helpful in slowing the ventricular rate.

Digitalis may be more effective if associated LV dysfunction is present; without such dysfunction, a beta blocker may be preferable to control the ventricular rate.

LONG-TERM MANAGEMENT [7]

For the rate control strategy, digitalis, calcium channel blockers (diltiazem and verapamil) and beta blockers can be used alone or in combination. For chronic management, digitalis is usually insufficient for adequate rate control during periods of exertion. One should not rely on a resting heart rate during an office visit as the sole evaluation of the adequacy of rate control. Ambulatory monitoring and/or exercise testing

can be useful to confirm adequate rate control during activity. In some patients with frequent recurrence and rapid ventricular rates not controlled by drugs or in

patients intolerant to drugs, modification or elimination of AV nodal conduction by radiofrequency catheter ablation and implantation of a rate-adaptive VVI pacemaker (VVIR) is acceptable rate control therapy. Whenever possible, atrial or dual-chamber pacing is preferable, because the incidence of atrial fibrillation and stroke appears to be reduced compared with VVI pacing. If a decision to maintain sinus rhythm has been made, class IA, IC, and III (amiodarone, sotalol) agents can be used to terminate acute-onset atrial fibrillation and prevent recurrences of atrial fibrillation. No one drug appears clearly superior, and selection is often based on the side effect profile and risk of proarrhythmia. This appears to be true for newer drugs such as azimilide and dofetilide as well, although comparative trials have not yet been done. Most antiarrhythmic drugs increase the likelihood of maintaining sinus rhythm from about 30 to 50 percent to 50 to 70 percent of patients per year after cardioversion. Some rather recent data suggest that sotalol is less effective than amiodarone or 1C agents. Class IA agents are often poorly tolerated and are rarely used in the long-term management of atrial fibrillation. As a

general rule, for patients without structural heart disease, IC agents (propafenone and flecainide) are well-tolerated and reasonable first-line agents for maintenance of sinus rhythm. For patients with structural heart disease, amiodarone, dofetilide, or sotalol may be reasonable first-line therapies to maintain sinus rhythm. Occasionally, these agents (especially class IC agents) can “convert” the atrial fibrillation to atrial flutter, which can be slower than usual because of the drug. In these patients, a strategy of an atrial flutter ablation (see later) and continuation of the antiarrhythmic drug can be effective for

maintaining sinus rhythm, with up to an 80 percent efficacy. Before electrical cardioversion, an antiarrhythmic agent is often administered for a few days to help prevent relapse of atrial fibrillation, as well as to convert some patients to sinus rhythm. In patients with occasional episodes of persistent atrial fibrillation (requiring cardioversion), a "pill in the pocket" approach can be effective. In these patients, either flecainide or propafenone, usually at high doses, is administered only after the onset of atrial fibrillation to convert the atrial fibrillation. For those patients who fail drug therapy, catheter ablation approaches have been applied successfully. Radiofrequency ablation aimed at electrical isolation of the pulmonary veins or wide area ablation of the posterior left atrium around the pulmonary veins is 70 to 85 percent effective in patients with paroxysmal atrial fibrillation and 50 to 70 percent effective in patients with persistent atrial fibrillation in short-term follow-up studies. The surgical maze procedure has been used to eliminate atrial fibrillation, particularly in combination with valve surgery, with a high success rate. Newer surgical techniques using specially designed tools (e.g., radiofrequency [RF] clamp, cryoablation, RF pen, high-intensity ultrasound) to replace the traditional cut-and-sew maze procedure enable the procedure to be done faster and through more limited incisions.

PATIENTS AND METHODS

INCLUSION CRITERIA

159 Patients who came to our cardiology out patient department between April 2008 and March 2010 were randomly selected and included in the study.

EXCLUSION CRITERIA

Patients with history of AF, atrial flutter, atrial tachycardia, age <18 years, previous pacemaker implantation or an implantable cardioverter-defibrillator were excluded from the study.

The following parameters were studied

- Age
- Sex
- Occupation
- Socioeconomic status
- Diabetes
- Hypertension
- Coronary artery disease
- Rheumatic heart disease
- Underlying disease like chronic obstructive pulmonary disease (COPD), thromboembolic events, heart failure, myopathies etc were studied.
- Medication use (beta blockers, verapamil, diuretics, anti arrhythmic etc)
- ECG
    1. Heart rate(bpm)

2. P-wave duration
3. PR interval
4. QRS duration (ms))

- Tissue Doppler imaging
    1. PA-TDI interval - (The time from the initiation of the P wave on the ECG (lead II) to the peak of A' wave on the lateral left atrial tissue Doppler tracing obtained in apical 4-chamber view) was measured
    2. Maximal A' –wave velocity (cm/s)(lateral left atrial tissue Doppler tracing)
- Dimensions (Echo cardiography)
    1. Aorta diameter(mm)
    2. Left atrial dimension(mm)
    3. Left ventricular end diastolic diameter(mm)
    4. Left ventricular end systolic diameter(mm)
    5. Inter ventricular septum width (mm)
    6. Posterior wall width(mm)
    7. Left ventricular mass(gm)
    8. End diastolic volume(ml)
    9. End systolic volume(ml)
    10. Caval vein(mm)
- Left ventricular function
    1. Left ventricular ejection fraction (%)

2. Hypokinesia

- Mitral valve Doppler assessments
    1. Maximal E – wave velocity (m/s)
    2. E – wave deceleration time (ms)
    3. Maximal A – wave velocity (m/s)
    4. E/A ratio
- Valvular disorders were studied.

TOTAL ATRIAL CONDUCTION TIME (TACT):

TACT is the time elapsed between the initiation of atrial depolarisation and the last depolarisation of the same activation front and it incorporates both conduction slowing and atrial dilatation

TACT is measured non-invasively by transthoracic tissue Doppler imaging of the Atria by measuring the PA-TDI interval.

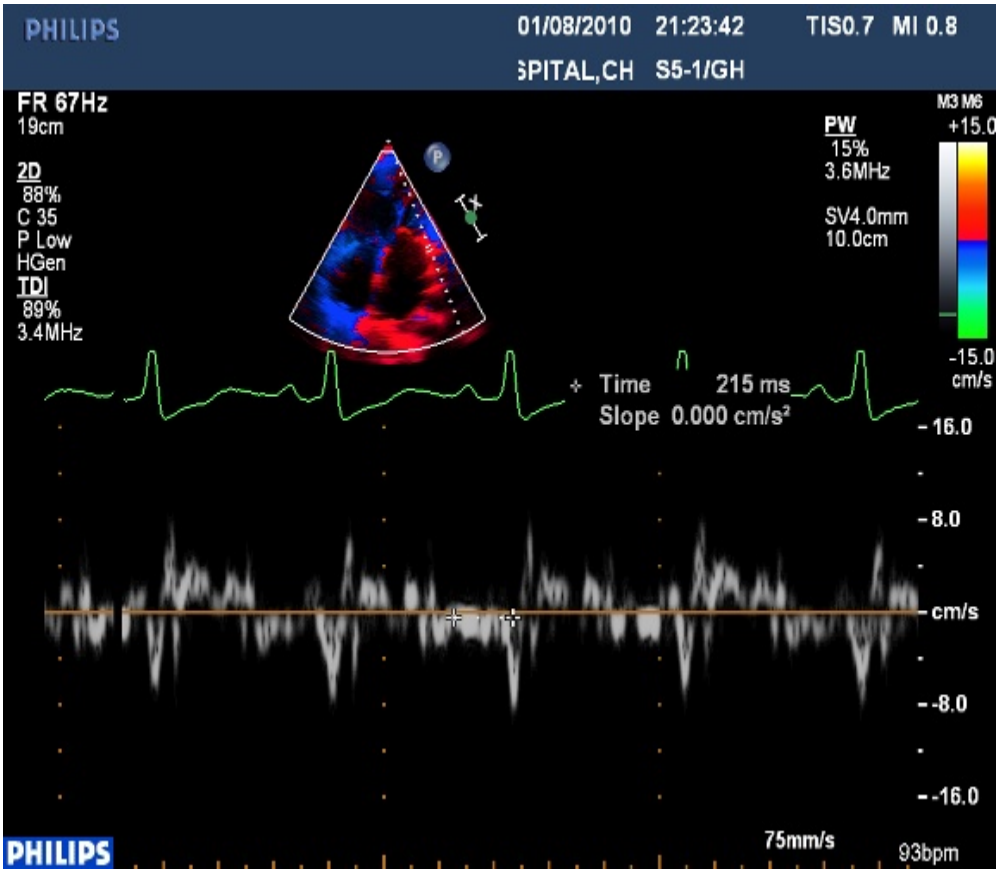


FIGURE 5 Shows the measurement of PA-TDI interval by tissue Doppler imaging

PA-TDI interval (the time from the initiation of the P wave on the ECG (lead II) to the peak of A' wave on the lateral left atrial tissue Doppler tracing obtained in apical 4-chamber view

- The measurements were made using Philips IE 33 ECHO Machine.
- Patients were followed up in the review OPD once in 2 months and rhythm Recorded by taking ECG.
- If any doubt holter recordings were done.
- Patients were advised to report to us whenever there is palpitation or other signs Of arrhythmia.

STATISTICAL ANALYSIS

- Continuous variables are reported as mean (SD) and categorical variables as observed number of patients (%).
- Cox regression univariate analysis used to compare patients who developed and not developed AF.
- Fisher' s exact test used to measure p value.

RESULTS

- The average age of the patients was 52.2 yrs,
- Male to female ratio was 1.5:1,
- Most of them belonged to low socioeconomic status,
- 20 patients (12 %) developed new-onset AF.
- These patients had a longer PA-TDI interval than patients who remained in sinus rhythm (176 ms vs. 148 ms, $p < 0.05$).

TABLE -2 PATIENT CHARACTERISTICS WERE SHOWN BELOW

SI NO	CHARACTERISTICS	ALL PATIENTS (N=159)	NO AF DURING FOLLOW-UP (N=139)	AF DURING FOLLOW-UP (N=20)	P VALUE
1	HYPERTENSION	95(60%)	87(63%)	8(40%)	0.0857
2	CAD	16(10%)	15(11%)	1(5%)	0.6954
3	DIABETES	20(13%)	18(13%)	2(10%)	1.0
4	THROMBOEMBOLIC EVENTS	8(5%)	7(5%)	1(5%)	1.0
5	RHD	70(44%)	60(43%)	10(50%)	0.6335
6	HEART FAILURE	14(8%)	10(7%)	4(20%)	0.0795
7	COPD	60(38%)	56(37%)	4(20%)	0.0899
8	MYOPATHIES	10(6%)	8(6%)	2(10%)	0.6156

1. The above table shows patient characteristics with predominantly hypertension, rheumatic heart disease, and chronic obstructive pulmonary disese(COPD).
2. Among the patients with myopathies (total no of patients-10), restrictive cardiomyopathy were-2, hypertropic obstructive cardiomyopathy-2, dilated cardiomyopathy were 6.
3. Some of the patients who were hypertensive also had diabetes and (COPD). Among them some had heart failure and thromboembolic events which is explained in the table above.

4. Age, diabetes, hypertension, Coronary artery disease (CAD), thromboembolic events, Rheumatic heart disease(RHD), Heart failure, Chronic obstructive pulmonary disease (COPD), myopathies and medication use were comparable between the groups which developed & not developed atrial fibrillation.

5. Maximal A' –wave velocity (cm/s) (lateral left atrial tissue Doppler tracing) were Rheumatic heart disease (total no of patients-70),mean maximal A'-wave velocity is 8.4 cm/s, restrictive cardiomyopathy(total no of patients -2)-7.1 cm/s, systemic hypertension(total no of patients -95) – 8.9 cm/s, hypertropic obstructive cardiomyopathy(total no of patients -2)-7.5 cm/s, dilated cardiomyopathy (total no of patients-6)-7.4 cm/s, patients who had systemic thromboembolism(total no of patients-8)-9.3 cm/s, chronic obstructive pulmonary disease(total no of patients-60) – 9.5 cm/s.
6. Left atrial diameter(mean) measured by 2d echo were, rheumatic heart disease-4.46cm, restrictive cardiomyopathy-5.9 cm, hypertropic obstructive cardiomyopathy- 3.25cm, systemic hypertension-3.8cm, patients who had systemic thromboembolism-4.6 cm, dilated cardiomyopathy-5.25 cm.
7. Left ventricular ejection fraction (mean %) measured (m-mode) were rheumatic heart disease-55.1%, restrictive cardiomyopathy-61.5%, hypertropic obstructive cardiomyopathy-65%, dilated cardiomyopathy-31.8%, systemic hypertension-57.9%, patients who had heart failure-48%.
8. Patients who were hypertensive had mean left ventricular posterior wall thickness – 10.4cms, and mean interventricular septal thickness-10.71cms,

hypertropic obstructive cardiomyopathy mean left ventricular posterior wall thickness-11.5cm, mean interventricular septal thickness- 26.5cms.

9. Mitral valve Doppler measurements, Maximal E – wave velocity (m/s) and Maximal A – wave velocity (m/s) (mean values) were rheumatic heart disease-0.55&0.38 m/s, restrictive cardiomyopathy -1.5&0.5 m/s, hypertropic obstructive cardiomyopathy- 0.35& 0.95 m/s, dilated cardiomyopathy-1.4&0.73 m/s, systemic hypertension-0.68&0.74 m/s, and patients who had coronary heart disease – 0.63&0.72m/s respectively.
10. E/A ratio(mean) were rheumatic heart disease -1.3, restrictive cardiomyopathy-3, hypertropic obstructive cardiomyopathy -0.7, dilated cardiomyopathy-2.03, systemic hypertension-0.91, and patients who had coronary heart disease – 1.01.

11. New onset AF was more common in patients with rheumatic heart disease (50%) (Especially with mitral regurgitation), chronic obstructive pulmonary disease (20%), restrictive cardiomyopathy (10%) and patients with previous history of Heart failure (20%). These findings are explained by the table below.

TABLE 3

PATIENTS WHO DEVELOPED ATRIAL FIBRILLATION (N=20)

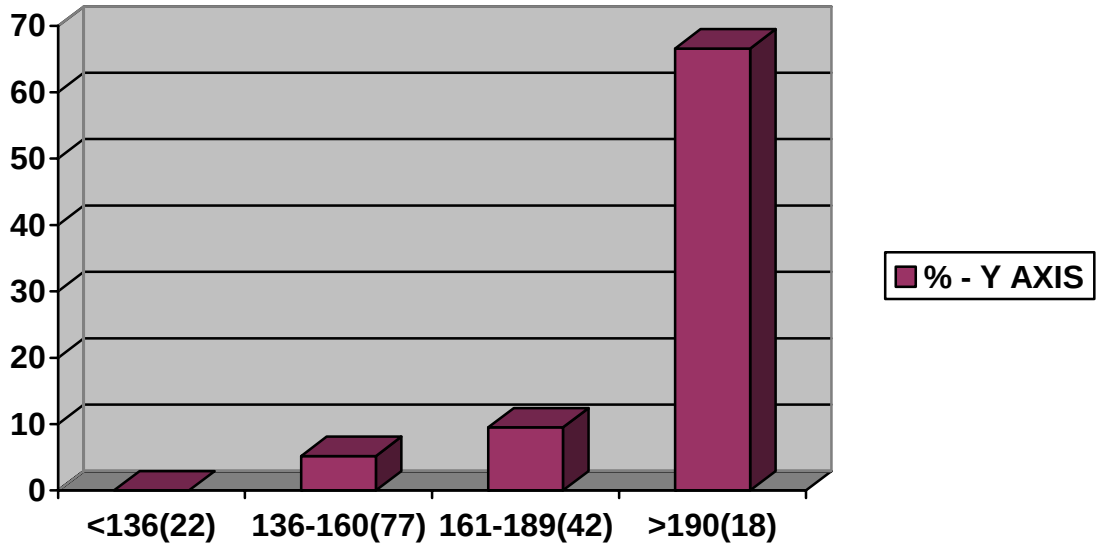
SI NO	DISEASE	NO (%)
1	RHD	10 (50%)
2	COPD	4 (20%)
3	RESTRICTIVE CARDIOMYOPATHY	2 (10%)
4	PRIOR CHF	4 (20%)

- Potential confounders were adjusted and it was found that prolonged PA-TDI was independently associated with new-onset Atrial Fibrillation (AF).
- With a PA-TDI interval <136 ms (n = 22), ($p < 0.05$) none had AF.
- With a PA-TDI interval >190 ms (n = 18), 12 (66%) had AF.
- With a PA-TDI interval 136-160 ms (n = 77), 4 (5.2%) had AF.
- With a PA-TDI interval 161-189 ms (n = 42), 4 (9.5%) had AF.

The following diagram explains these findings.

FIGURE 6

INCIDENCE OF NEW-ONSET ATRIAL FIBRILLATION (AF) IN PATIENTS CLASSIFIED ACCORDING TO THE PA-TDI INTERVAL MEASURED AT BASELINE



PA-TDI Interval in ms (NO of patients)

Cox regression showed that PA-TDI was independently associated with new-onset AF (OR=1.370; 95% CI 1.032 to 1.723; p=0.028).

Thus the PA – TDI interval independently predict the new onset atrial fibrillation.

DISCUSSION

The hemodynamic function of the left atrium (LA) [3] primarily modulates the left ventricular (LV) filling through its three components: a reservoir component during ventricular systole, a conduit component during early ventricular diastole, and a booster pump component during late ventricular diastole. Left atrial appendage (LAA) is a highly contractile structure with a pattern of contractions totally different from that of the LA main body. It is more compliant and therefore plays an important role in the LA reservoir function, especially during increases in the LA pressure or volume

Atrial fibrillation (AF) is the most common cardiac arrhythmia associated with an at least two-fold increase in morbidity and mortality, and occurs in 0.4% of the general population, increasing to 5% in those >65 years old. With the loss of atrial booster pump function, the LA–LV pressure gradient during early LV filling is enhanced by elevation of the LA pressure to maintain stroke volume. Thus, a reduction in both LA compliance and volume has been observed with the onset of AF that further decreases cardiac function and increases the risk of thrombo-embolism.

Importance of predicting atrial fibrillation:

Atrial fibrillation [3] constitutes the commonest cardiac rhythm disorder and is the major determinant of outcome in valvular, myocardial, and ischemic heart disorders.

AF can result in life-threatening complications such as stroke and heart failure. Therefore, the application of adequate individual treatment is essential. Unfortunately, many patients have silent AF. As a result, treatment may come too late. Adequate administration of oral anticoagulation will reduce the risk of thrombo-embolic complications by 60%. Therefore, one could imagine that prophylactic anticoagulation

could be applied in patients with a high risk of developing AF. However, this hypothesis needs to be confirmed in prospective randomized trials.

Observational population[1] studies such as the Framingham Heart study, the Manitoba Follow-up study and the Cardiovascular Health study reported several clinical and echocardiographic parameters that are associated with the development of new onset AF. The clinical factors associated with new-onset AF are higher age, male sex, the presence of diabetes, hypertension, congestive heart failure, valve disease and coronary artery disease.

In our study, new onset AF was more common in patients with rheumatic heart disease (50%) (especially with mitral regurgitation), chronic obstructive pulmonary disease (20%), restrictive cardiomyopathy (10%), and patients with previous history of heart failure (20%).

Atrial electrical activation[2], as assessed by the PA-start interval, began at the RA and followed through the IAS, to the inferior and posterior LA walls. This is the known normal electrical activation process, as obtained by invasive electrophysiology techniques.

The atrial fibrillation cycle length (AFCL) is a critical parameter for the perpetuation and termination of AF. Currently, the total atrial activation time[1], as indicated by the P-wave duration using signal-averaged (SA) electrocardiogram (ECG) (SA-ECG), is the most powerful predictor of atrial fibrillation. However, because of practical limitations, this technique is not used in clinical routine.

Left atrial enlargement, increased left ventricular wall thickness[1], left ventricular diastolic dysfunction and a reduced left ventricular fractional shortening are predictive

echocardiographic parameters for new-onset AF. There is no simple tool available to predict the onset this arrhythmia. Electrophysiological parameters are too complex and can not be obtained in bedside.

The identification of an abnormal electrical activation process could be of interest in some patients with atrial fibrillation or other supra-ventricular tachy-arrhythmias, in whom the premature atrial contraction acting as a triggering factor could be aggravated by local delayed conduction. Several studies have shown the independent prognostic value of atrial function measurements in subsets of patients.

Karl et al[12], even as early as in 1978 in an article published by the AHA in Circulation showed that prolonged atrial conduction was a major predisposing factor for the development of Atrial flutter.

The multiple wavelet re-entry theory[1] postulated the existence of multiple spatially discrete activation fronts (wavelets) resulting in re-entry at changing locations as the basis for AF. A decreased conduction velocity, leads to a shorter wavelength of the reentrant wave fronts. An increased atrial size can harbour more wave fronts of a certain size at the same time. Both will favour AF.

The change of the LA function in different phases can be assessed non-invasively by echocardiography, using conventional methods such as changes in LA area and volume, but they lack specificity.

Novel techniques such as tissue Doppler imaging (TDI) TVE-derived variables of atrial mechanical function may have an additional role for facilitating the assessment of atrial function and consequently in the process of risk stratification. Tissue Doppler

imaging can be used to measure total atrial conduction time. A prolonged PA-TDI interval may predict the development of new-onset AF.

De Vos et al[1] showed that an increased Total Atrial Conduction Time, which is the time elapsed between the initiation of atrial depolarization and the last depolarization of the same activation front, incorporates both conduction slowing and atrial dilatation, and may therefore reflect the existence of a substrate vulnerable for AF. Determination of the TACT may therefore be better than the classical predictors of AF since it might not only demonstrate the presence of underlying disease but also its severity. Thus, different LA walls with their corresponding levels from the mitral annulus can be compared and assessed, in particular by offline analysis of colour TDI, such as septal and lateral walls in an apical four-chamber view, anterior and inferior walls in a two-chamber view. The atrial myocardial velocity curve consists of three major deflections: ventricular systolic (Sa), early ventricular diastolic (Ea), and late ventricular diastolic (atrial contraction, Aa) waves. (The Aa-wave has been regarded as a direct measure of regional active atrial contraction on the longitudinal axis, which might be less load dependent. The Sa and Ea waves may represent the passive expansion and emptying components of the LA function. Approaching regional left atrial function by tissue Doppler velocity and strain imaging were described very early by Qing Zhang et al[3].

In our study, TDI was done by placing a 2mm sample volume PWD at the lateral wall of the left atrium, just below the mitral annulus.

P-wave duration on the 12-lead ECG and A' wave using the mitral Doppler flow signal provide estimates of the TACT. PA-TDI[1] determined by transthoracic tissue Doppler imaging had the best correlation with the ''gold standard'' (SA-ECG P-wave

duration). Previous studies presented a similar parameter measuring the interval between the onset of the P wave on the ECG till the onset of the A' wave of the left atrial lateral wall instead of the peak of the A' wave (atrial electromechanical interval or AEMI). But, studies done later proved that the peak of the A' wave is a better pointer than the onset of the wave. In our study, the peak of the A' was used and the distance in milliseconds from the onset of the 'p' wave of the ECG was used.

The main new findings of this study[2] are as follows:

(1) Some TVE-derived variables indirectly reflect the atrial electrical activation that follows the known activation process as revealed by invasive electrophysiology.

(2) The regional and global atrial mechanical function is explained by an upward movement of the atrial walls at the region near the A-V ring with a continuous reduction of this movement towards the upper levels of atrial walls.

(3) The atrial mechanical function is quite similar in all LA walls; however, all indices of mechanical function were higher in the RA than in the LA. The difference in the atrial velocities at different sites was attributed to an atrial free-wall motion higher than that of the bounded IAS. Furthermore, the larger pectinate muscle in the RA can perhaps generate a more pronounced and sustained longitudinal movement in the relatively low pressure system of the right ventricle.

(4) The present study showed that all atrial walls actively moved upwards from the region of the A-V ring at late diastole, with a reduction of this movement towards the upper parts, thus empting the atria and contributing towards the last part of filling of the LV. This longitudinal movement of the atrial walls is probably related to the longitudinal endocardial muscular fibers along the walls of the LA and RA. The more pronounced

longitudinal movement in the RA may be explained in part by the larger pectinate muscles in the RA, but also by the lower pressures in the heart's right side. To what extent circumferential contraction of the atrial muscle fibers might contribute to atrial mechanical function is unknown. Anatomically, the large amount of circumferential muscle fibers present in the vestibules of the RA and LA might imply some kind of circumferential or radial contraction of the atria.

(5) No correlations were found between 2-D- and TVE derived variables of atrial mechanical function, as was also found in a previous study by Donald et al. Although 2-D-derived variables measure volumes and volume-derived indices that might indicate some kind of atrial mechanical force, it was surprising to find no correlations between the variables obtained by the two different techniques. This might indicate that the velocities and the displacements registered from all atrial walls by TVE are less dependent on volume loading conditions than 2D derived variables and therefore could be used as reliable measurement of pure atrial mechanical contraction or inotropism. However, most of the TVE-derived variables expressing atrial mechanical function had good values of repeatability and measurement error. Assessing atrial mechanical function by measuring volumes is time consuming and depends on age, gender, and body surface area. In addition, atrial volume indices are also dependent on loading conditions and are not necessarily more reproducible than TVE-derived variables.

Our study demonstrates that the TACT as determined by tissue Doppler imaging may help to identify patients with a substrate vulnerable for AF. After correcting for possible confounders, the PA-TDI interval remains the most important predictor of new-onset AF. The longer the PA-TDI interval, the higher the incidence of new-onset AF.

Therefore, the PATDI interval may become a useful measure for risk stratification to improve efficiency of primary prevention of AF.

In a study by De Vos et al[1], they demonstrate that a short PA-TDI interval of <130 ms seems to prevent patients from developing AF. Therefore, one could hypothesize that these patients are not candidates for primary prevention. Patients with a PA-TDI interval of >165ms have a reasonable chance of developing AF and could be candidates for primary prevention using ‘‘upstream’’ cardiovascular drugs. A PA-TDI interval 190 ms makes patients very vulnerable for the development of AF. These patients could be treated with prophylactic anticoagulation.

In our study, 20 patients (12 %) developed new-onset AF. These patients had a longer PA-TDI interval than patients who remained in sinus rhythm (176ms vs. 148 ms, $p < 0.05$). With a PA-TDI interval <136 ms, none had AF ($p < 0.05$).

Other uses of measurement of total atrial conduction time:

The assessment of pure mechanical atrial function by means of atrial wall movements may give more concrete clues about the recovery process of atrial electromechanical function after conversion for atrial fibrillation and flutter and can give additional patho-physiological insights on the thrombo-embolic process that occur in some of those patients. TVE-derived parameters may also give additional patho-physiological information on the process of atrial electromechanical remodeling that occurs in patients with sustained supra-ventricular tachy-arrhythmias

Ischemic heart disease[3]:

Atrial contractile dysfunction appears early in ischemic heart disease (IHD), irrespective of previous myocardial infarction, co-existing systolic dysfunction, or severity of diastolic dysfunction. Yu et al, found that the VA' measured at mid-level of the IAS and the lateral LA in the apical four chamber view were drastically reduced in 118 patients with IHD when compared with 100 normal subjects. A poor LV-ejection fraction and the presence of a restrictive LV filling pattern were the most important determinants of LA contractile dysfunction in IHD.

In our study, 10% of our patients had Coronary artery disease, and a significant number developed AF during follow-up.

Advanced heart failure and cardiac resynchronization therapy[3]:

Gabriel et al showed that the LA mechanical function can be modified by heart failure treatment, such as cardiac resynchronization therapy (CRT), which is of proven benefit to advanced heart failure patients with prolonged QRS duration.

In this study, 8% of the study patients were included as they had heart failure. 20% of those patients developed AF during follow-up. Even these patients might improve with CRT therapy.

Mitral stenosis and mitral regurgitation[3]:

Due to inflow obstruction, the atrial booster pump contributes less to LV filling in mitral stenosis even during sinus rhythm, despite a proportional increase, with increasing severity, in the LA preload. The impaired atrial reservoir and pump function are associated with a reduction in LA compliance and intrinsic myocardial contractility. In addition, the LAA is inevitably affected in mitral stenosis with reduced contribution to

overall LA emptying fraction and increased risk of thrombus formation. In a study by Wang et al, published in 2005, the LSW and LDW tissue velocities recorded during sinus rhythm at the lateral wall or at the tip of the LAA were markedly reduced in subjects with mitral stenosis when compared with normal subjects. Systolic velocity was further decreased in patients with spontaneous echo contrast (SEC) in the LAA than those without. In this present study, the bulk of the patients were suffering from RHD (44%) and the incidence of atrial fibrillation during follow-up was also high in those patients, in concordance with the previous studies (50% developed new onset AF).

LIMITATIONS

- Study was not compared with electro physiological measurements.

For the future:

Further refinement of the TVE technique are necessary not only to identify the mechanical activation atrial sequence during normal sinus rhythm, but also to identify the origin and the activation sequence of supra-ventricular ectopic beats and in patients with RA, IAS or bi-atrial pacing. Our study was first of its kind in our region and needs to be compared with electrophysiological measurements. Thus, TVE could be an excellent adjunct to invasive electrophysiological techniques in selecting adequate patients and in the evaluation of atrial electromechanical consequences of RA, IAS or bi-atrial pacing.

CONCLUSIONS

- **Tissue Doppler imaging can be used to measure total atrial conduction time.**
- **A prolonged PA-TDI interval may predict the development of new-onset AF.**

BIBLIOGRAPHY

1. Atrial tissue Doppler imaging for prediction of new onset atrial fibrillation,

C B De Vos et al, Heart 2009; 95:835–840.

2. Assessment of atrial regional and global electromechanical function by tissue velocity echocardiography: a feasibility study on healthy individuals

Quintana M, Lindell P, Saha SK, del Furia F, Lind B, Govind S, Brodin L

Cardiovascular Ultrasound 2005, 3:4 (18 February 2005)

3. Approaching regional left atrial function by tissue Doppler velocity and strain imaging Qing zhang et al, Oxford Journals MedicineEP Europace Lvolume 10 supplement 3 Pp. iii62-iii69

4. Intra-Left Atrial Mechanical Delay Detected by Tissue Doppler Echocardiography Can Be a Useful Marker for Paroxysmal Atrial Fibrillation Deniz, Ali et al Echocardiography - Jnl Cardiovascular Ultrasound & Allied Techniques, Volume 26, Number 7, August 2009 , pp. 779-784(6)

5.Assessment of Atrial Conduction in Patients with Scleroderma by Tissue Doppler Echocardiography and P Wave Dispersion

Ilknur Cana, Ahmet Mesut Onatc, Kudret Aytemira, Ali Akdoganb, Kemal Uretenb, Sedat Kirazb, Ihsan Ertenlib, Necla Ozera, Lale Tokgozoglua, Ali

Cardiology 2007;108:317-321

6. Interatrial dyssynchrony on tissue Doppler imaging predicts progression to chronic atrial fibrillation in patients with non-valvular paroxysmal atrial fibrillation.Sakabe K, Fukuda N, Fukuda Y, Morishita S, Shinohara H, Tamura Y. Heart. 2009 Jun;95(12):988-93. Epub 2009 Mar 5.

7. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine, 8th ed.

8. The Echo Manual, 3rd Edition Oh, Jae K.; Seward, James B.; Tajik, A. Jamil

9. Feigenbaum's Echocardiography, 6th Edition, Feigenbaum, Harvey; Armstrong, William F.; Ryan, Thomas

10.Helle-Valle T, Crosby J, Edvardsen T, et al: New noninvasive method for assessment of left ventricular rotation: speckle tracking echocardiography. Circulation 112:3149, 2005.

11.Hurst's The Heart, 12th Edition

12. Karl et al , Circulation vol 57, no 2, february 1978

13. Assessment of regional atrial function in patients with hypertrophic cardiomyopathies using tissue Doppler imaging.
Telagh R, Hui W, Abd El Rahman M, Berger F, Lange PE, Abdul-Khaliq H.
Pediatr Cardiol. 2008 Mar;29(2):301-8.

14. Clinical utility of tissue Doppler imaging in prediction of atrial fibrillation after coronary artery bypass grafting.
Benedetto U, Melina G, Roscitano A, Ciavarella GM, Tonelli E, Sinatra R.
Ann Thorac Surg. 2007 Jan;83(1):83-8.

15. Pictorial representation of atrial depolarization on the basis of dipole electrocardiotopography (decarto) for diagnosis of atrial enlargement
Advances in electrocardiology 2004 (pp 644-647) author(s):l.i. titomir

16. Advances in electrocardiology 2004 (pp 512-516) author(s): raija koskinen

GLOSSARY AND ACRONYMS

AF- Atrial fibrillation

COPD-chronic obstuctive pulmonary disease

CAD- Coronary artery disease

HOCM-Hypertrophic obstructive cardiomyopathy

RHD-Rheumatic Heart disease

TDI- Tissue doppler imaging

PROFORMA

Age Sex Occupation date

RISK FACTORS

Diabetes Hypertension Coronary artery disease

Rheumatic heart disease chronic obstructive pulmonary disease (COPD), thromboembolic events, heart failure, myopathies.

Medication use

ECG

Heart rate(bpm)

P-wave duration

PR interval

QRS duration (ms)

Tissue Doppler imaging

PA-TDI interval

Maximal A' –wave velocity (cm/s)

Dimensions (Echo cardiography)

Aorta diameter(mm)

Left atrial dimension(mm)

Left ventricular end diastolic diameter(mm)

Left ventricular end systolic diameter(mm)

Inter ventricular septum width (mm)

Posterior wall width(mm)

Left ventricular mass(gm)

End diastolic volume(ml)

End systolic volume(ml)

Caval vein(mm)

Left ventricular function

Left ventricular ejection fraction (%)

Hypokinesia

Mitral valve Doppler assessments

Maximal E – wave velocity (m/s)

E – wave deceleration time (ms)

Maximal A – wave velocity (m/s)

E/A ratio

Valvular disorders - DETAILS